GENERAL PARESIS

A CLINICAL LECTURE

BY SMITH ELY JELLIFFE, M.D., PH.D.

Clinical Professor of Mental Diseases, Fordham University, New York;
Visiting Neurologist, City Hospital, New York; Senior Physician,
New York Hospital for Nervous Diseases

Reprinted from International Clinics, Vol. III., Eighteenth Series.

GENERAL PARESIS

A CLINICAL LECTURE

BY SMITH ELY JELLIFFE, M.D., PH.D.

Clinical Professor of Mental Diseases, Fordham University, New York; Visiting Neurologist, City Hospital, New York; Senior Physician, New York Hospital for Nervous Diseases

---

I DESIRE to call your attention to a form of mental disorder which represents one of the most clearly defined types of mental disease with which we are acquainted, and yet, at the same time, one which in its different phases calls for no mean exercise of critical acumen, namely, general paresis, or the much poorer term which should be abandoned, general paralysis of the insane. This has the lay name "softening of the brain" which term, however, is used, as we shall see later, to designate more than one condition.

I have pointed out to you at frequent intervals the gradual subdivisions, which are termed classifications, that advancing studies have made in the disorders of the mind. You will recall that of these classifications that of febrile deliria constituted from the very beginning a series of stages which were recognized to be distinct from other forms of mental disorder. It was not unknown to the ancients, however, that certain forms of dementia were associated with paralysis, but it is uncertain just when it was appreciated that the motor defect was a necessary part of the mental disease.* It may be of interest to note that the word παραλύσις

---

* Thus Celsus (30 A.D. *circa*), in the 26th chapter of his third book, which in the main is devoted to the disorders of the mind, speaks of a form of mental disorder which he says "with us, occurs but seldom. The body and the mind are dull, paralytic strokes occur at times, and again at other times there is a disturbance from other disease." Flemming has suggested that Celsus for lack of a better place to put this form arranged it with the apoplexies, and in all our English translations the real relations of Celsus' ideas are lost. Thus Grier considers the apoplexy the important feature and so arranges the translation, but in Almevoleen and Lesart, the idea here given is set forth. Celsus says the Greeks called it apoplexy, but he has put it in his insanities. Whether aphasia, or paresis is meant it is not yet possible to decide.

used by the ancients really means dissolution, and we are not sure that this disorder may not have been in mind. In fact, even at the present time, you will find a school of psychiatrists who deny that general paralysis is a special disease type, but who consider that the paresis is only an etiological factor, and that the symptomatology may be designated by a vast variety of names. I shall recur to this at a later period. It is certain, however, that as early as 1670, Willis [1] distinguished a type of mental disease, which was associated with mental paralysis, as follows: "I have observed in many cases that with their cerebrum out of order to begin with, they became affected by a dulness of mind, and loss of memory, followed by a condition of stupidity and idiocy, and finally developed a paralysis—a succession of events that I used to be able to forecast," and other English authors, notably Haslam and Perfect, described conditions which we recognize at the present time as true cases of paresis. Thus, in 1788, Haslam wrote, in his Observations on Madness and Melancholy, "Paralytic affections are a common cause of madness, as men have believed, and are also a common result of mania. Paralytics, as a rule, show disturbances of motion which are absolutely independent of their mental affection. The speech is disturbed, the angles of the mouth are retarded, arms and legs are robbed of their natural motion, and in the majority of cases the memory is greatly weakened. The consciousness of their positions is lost by these patients as a rule. Although so weak that they can hardly hold themselves up on their legs, they nevertheless believe themselves to be strong, and able for the greatest of endeavors."

Esquirol, some eight years after Haslam, namely, in 1796, described the speech difficulties occurring in certain mental disorders, but this noted psychiatrist did not differentiate paresis from other allied psychoses. You will find that most of the historical sketches of this disorder ascribe to Bayle the distinction of having first separated this form of disease from others. And it may in truth be said that Bayle's general conceptions were the most fruitful in developing the ideas which later led to the formation of paresis as a more or less distinct type.

---

[1] Anima Brutorum.

Bayle, in his thesis l'Arachnitis chronique, 1822, and in his Traité des maladies du cerveau et des ces membres, 1826, established the autonomy of many of the different symptom groups, and sought to bring the whole disorder into line—divergencies from which point of view have been frequent even up to present times. We have seen that Willis says that from certain mental symptoms he could prognosticate the coming on of the paralytic signs, and it is not difficult to read much into his short statement already quoted. But dualistic interpretations up to the time of Bayle were the rule rather than the exception, and the other symptoms considered complications rather than being a part of the disease. Baillanger represented this clinical tendency which has its modern imitators, notably in certain English circles, and we read in Baillanger of excited cases being called folie paralytique, and of demented cases being called demence paralytique, the two being different diseases.

Bayle, 1823, and Calmeil, 1829, called particular attention to the changes in the pia which have passed current as synonymous with paresis to the present time although such changes had been recorded and correctly interpreted, even if their clinical alliances were not understood.

Esquirol, as early as 1814, called attention to the speech disorders, Georget in 1823 to the epileptiform attacks, and Calmeil in 1826 brought together our first monograph on the disease, in which he taught it to be a disease *sui generis,* without any specific pathological anatomical changes, but associated with the appearance of a chronic inflammation of the pia (cloudiness, thickening, attachment to the cortex, dilatation of its veins) and of the cortex. He recognized the excited agitated types, the depressed or melancholic types, and the simple dementing types, and by 1841 at least, the unification of psychic and motor disturbances was taught by him in a decided manner. We have indicated the false teachings of Baillanger, which were later controverted by Falret, who in a monograph in 1853, gave a masterly outline of the disease largely as we understand it to-day. Duchen, in 1851, was the first German author to take this same stand.

Joffe in 1857, and Westphal, first called attention to the degeneration in the posterior columns, and started the discussion of the

relation of tabes to paresis so well summarized in a recent study by Cotton ("American Journal of Insanity," 1905).

The most important monographs are those of Voisin in 1879, Mendel in 1880, Mickle in 1886, Chase, 1902, and Kraft Ebbing and Obersteiner, 1908. The text book descriptions of Dupré in Ballet's Traité, and of Kraepelin's in the seventh edition of his text book, 1904, are the most exhaustive, while from the pathological point of view the study of Alzheimer, 1904, in Nissl's "Histologische Arbeiten, Vol. I, will remain the fundamental classic in the field for many years to come.

It seems to me desirable, at this time, that I should call your attention again to the question already raised as to what should be considered a type of a mental disease. That is, can mental diseases be thought of from precisely the same point of view as diseases of other organs of the body; or, shall we be compelled to follow the general idea which is so widely prevalent, especially among the laity, that there is practically but one disease of the mind, namely, insanity, and that which we recognize as types are only illy assorted symptom pictures. Judges only too frequently have this point of view as well, and the whole medico-legal terminology has not advanced beyond this narrow concept.

I desire to negative this latter view most emphatically, and it is at this time that it can be done most conveniently, for modern psychiatry recognizes in general paresis the prototype of a distinct disease process in much the same manner as we recognize a definite disease in other organs of the body. That is, we have advanced to such a point that we may say that, notwithstanding the varying symptom pictures we have in paresis a disease process, with a very definite underlying pathological foundation, which foundation admits, according to criteria established by Nissl and Alzheimer, of little contradiction. Given a patient, no matter what the confusion may be in the interpretation of the clinical picture, who shows the classical pathological changes, this then is a case of paresis, and it becomes the duty of psychiatrists to establish definite clinical criteria, by which it may be set apart from other similar combinations of mental and physical symptoms.

First a word as to the conditions in which the similarities of

clinical symptomatology may be present, and then let me pass to the necessary pathological foundations of this disorder.

It is now clearly established that a number of clinical pictures come to resemble general paresis very closely, and yet are due to an entirely different pathological foundation. It becomes the problem of the future to point out wherein, by a more exact clinical analysis, the minute variations which frequently exist, may be made more valuable diagnostically. These cases are those associated with arteriosclerosis in the senile and presenile states, with alcoholism, with cerebral syphilis, either of the diffuse variety or that type consisting of a combination of gummata with meningeal changes, with tuberculosis of the brain, with cerebral tumor, with diffuse gliosis, allied to multiple sclerosis, and with chronic pachymeningitis. These are the chief pathological conditions which are known to be present in the brains of some patients who have presented a clinical picture undifferentiable from general paresis, and were recognized not to be such only after death. The percentage of these unrecognizable cases (*i.e.*, only recognizable postmortem) has been variously estimated to be from five to ten per cent., but as most of these estimates have been formed before the days of the study of the cerebrospinal fluid for lymphocytes, and for syphilitic antibodies, it is highly probable that a new review of the statistics will reduce this estimate very considerably.

Pathology.—Before entering upon even a short sketch of the pathological features of general paresis, I desire to call your attention to the fact, that much of the easy talk that you hear, and even read in text books, whose authors ought to know better, that we know comparatively little of the pathology of mental disease, is pure buncum. If the authors should be honest and confess that they themselves had not taken the time and trouble to learn what is known, it would represent the truth, for there is an immense field, and it has been very faithfully studied, and the advances in technic are so many, and newly acquired, that it requires a very progressive worker to keep within sight of what is being done with the pathology of the brain in its relations to mental disorders. What is true, however, is that a recognized invariable pathology is not yet established for all of those disorders which we have considered to be clinical types of mental disease. Certainly the same thing has been

true in the study of tuberculosis, if one reads history aright, and it is only beginning to be appreciated how varying are the pathological pictures of the different types of tuberculosis of the lungs. The really efficient methods for studying nervous tissues have as yet been in use very few years compared with the years of progressive study of the comparatively simple lung tissue.

When I show you the Histological Study on the Differential Diagnosis of General Paresis by Alois Alzheimer in the first volume of Nissl's Histological and Pathological Studies of the Cortex, published in 1904, you will realize how difficult a task it is to concentrate in a short talk the features of so exhaustive and fundamental a research. I take it that no one here will hope really to know the problems presented by this disease without reading this work.

What are the main features that are present in the brain of a paretic, who has not died in the very early stages of some intercurrent trouble?

The gross appearances have been described for many years; bear in mind that they are not reliable. They are true for the majority of the cases, but not for all, either in the presence of negative signs, or even with positive signs. These are frequently put down as thickening of the bones, thinning of the diploe, thickening and clouding of the pia, with attachments to the cortex, hydrocephalus internus, externus, granulations of the ependyma, atrophy of the brain and of the cortex. Many a diagnosis of paresis is made on these findings. Such was the diagnosis in the case of Schumann, the musician, who probably suffered from dementia præcox and not paresis; but Alzheimer shows without doubt that all of these may be present, and yet the case be not one of paresis. Senile dementia, dementia præcox, syphilitic gummomeningitis, arteriosclerosis may give in cases any or all of these changes. Thus the diagnosis depends, to be absolute, on a histological examination.

The histological variations are present in the membranes, and in the cortex. No satisfactory histological differential features are found in the alterations of the bones.

The changes in the dura are not of great moment. In rare instances the vessels are infiltrated with lymphocytes and plasma cells. Those of the pia are more characteristic. Practically every

case of paresis shows changes in the pia. Here there is a marked infiltration thickening. There are marked changes in the blood vessels, consisting of a peri-arteritis and lymphocyte infiltration. Plasma cells are frequent, lying in and around the smaller vessels. Many show themselves in a degenerative condition. There is further a proliferation of new blood vessels, these extending into the cortex, with the greatly increased number of connective-tissue elements cause the adhesions to the cortex, formerly held to be alone diagnostic of paresis. Regressive changes in the blood vessels are also very frequent.

The changes in the cortex itself are of the utmost importance. These vary considerably in their intensity, and particularly in their locality, at times they are very diffuse, involving all classes of cortex structures, at other times localized to a comparatively restricted portion of the brain area. Following Alzheimer, we will look separately at the different structures involved, thus taking up in order the changes in I, vessels; II, ganglion cells; III, axis cylinders, and IV, glia.

I. *Blood Vessels.*—These changes have been very extensively studied, and Calmeil, as early as 1826, described the cell groupings about the vessels now known as lymphocytes, and plasma cells. These changes in the vessels are quite distinct from those observed in arteriosclerosis or hyaline degeneration.

a. One of the most striking features is the marked proliferation of new capillaries. These are often extremely rich, often showing like a thick net-work of capillary meshes. These are the product of an active, productive, inflammatory process, and come about through the very rich formation of new endothelial cells, and the branching and vascularization of the regenerating intima. This new vessel formation in some cases is excessive, in others less noticeable.

b. There results an increase of elastic tissues by the formation of new net-works about the proliferating endothelial cells, and a formation of stronger membranes.

c. A proliferation of the adventitia also results, at times slight, again very marked.

d. A dilatation and infiltration of the adventitial lymph-spaces. The infiltrating cells are largely made up of plasma-cells.

They are never absent even in the most acute case. Lymphocytes and mast cells are also common in the lymph-spaces.

e. In most of the advanced cases of paresis, degenerative changes are common in the blood vessels, especially in the upper cortical layers. A complete destruction of the vessels may take place and hyaline degeneration result.

f. In the cortex of the paretic, a peculiar cell form, Nissl's Stäbchen zell or rod-like cell, is uniformly present. Alzheimer thinks they develop from some of the elements of the blood vessels. Curiously enough Mott calls them collapsed capillaries.

II. *Ganglion Cell Changes.*—*a.* These are extremely diverse and widespread, but are not of themselves pathognomonic of paresis. Practically no case of paresis is known that does not show ganglion changes of some character.

b. These changes embrace practically all of the forms of ganglion-cell alteration that have been described. Many of these are extreme in grade, as shown by the rapid degeneration, and the necrobiotic changes that are present.

c. A great number of ganglion-cells are completely destroyed in the advanced cases.

d. Not only are the ganglion cells altered, but in all probability the finer nerve structures which lie between the ends of the sheath-covered axis cylinders and the ganglion cells are distinctly involved.

e. In the majority of advanced paretics, the arrangement of the ganglion-cell groups is modified, *i.e.,* the cell architecture of the cortex is changed in lesser or greater degree.

III. *Changes in Axis Cylinders.*—These undergo early degeneration in many cases. Present technical methods do not permit the demonstration of these alterations in the very early stages, but in the advanced cases, the degeneration of the axis cylinder is very marked, such breaking down either being localized or involving the entire cortex.

IV. *Changes in Glia.*—Changes in the connective-tissue elements play a very prominent rôle in the pathology of paresis. In older studies the glial elements were often taken for lymphocytes, and it has only been within recent years that the pathological alterations of the connective-tissue elements have received their proper

share of attention. Let me call your attention to the fact that in the study of this important issue the work of Bevan Lewis, an English psychiatrist, is very noteworthy.

a. In paresis there is a very marked growth of the connective-tissue elements. In the advanced stages such increase of glia elements forms almost a felt of this tissue in the cortex.

b. The major portion of the new glias are found on the outer layers of the cortex, making it stronger, as it were. The thickening due to new glia elements is particularly noticeable about the blood vessels.

These are the fundamental changes, but it is to be noted that the greatest variation exists as to the location of the major modifications in different individuals. Thus a changing clinical picture has its counterpart in an extremely variable pathological involvement. Variation of place of involvement, and time of involvement, with intensity and rapidity all modify the clinical course very markedly. It should further be borne in mind that not only is the cortex involved, but the basal ganglia, the cerebellum, the medulla, the pons and the spinal cord are subject to the exact type of pathological alteration that I have just described. Pathologists have a tendency to extend the limits of the pathology of paresis to other organs of the body.

With the possibilities then of such endless variety of pathological variation, it is not to be wondered at that so many clinical pictures are known, and how rapidly the clinical picture may vary from week to week and from month to month.

It is certain that pathological alterations may exist for some time without sufficient disturbance of function to create any symptomatology. Physical symptoms may precede the mental ones, or vice versa. The order of the symptoms will depend essentially on the site of earliest or worst involvement, and as our cases are studied more in detail, it becomes evident how rich the picture is. Thus, it is known that certain cases have begun with hallucinations of hearing. Accidental death before the development of other symptoms revealed changes typical of paresis confined almost to the temporal lobes. This instance shows how important it is to weigh every symptom and to realize the need for caution in approaching all mental cases.

Those patients who show a similar picture in the great majority of attacks have what we call classical paralytic dementia, but let us all bear in mind that statistical studies, while very useful in telling you what has happened, are of little service in the first case any one of you may see. You are very liable to see the non-classical cases, and to fail to recognize them.

In the majority of the cases which have been studied anatomically, it is found that the entire cortex is involved. The greatest involvements are found in the occipital lobes, the poles and the anterior ends of the convexity of the frontal lobes, the parietal lobes and the central convolutions. The amount of involvement of the temporal lobes seems to be subject to the greatest variation.

Paralytics with more or less well-marked local signs of spots of cerebral softening causing partial paralysis, aphasias, etc., belong to the rarer kinds met with, yet they do occur. Hoch has recently reported such a case in full. Alzheimer enters into the literature thoroughly.

Etiology.—We will not stop very long with the etiology. Let us assume its syphilitic nature. This seems to be the chief factor, not so much from the statistical enquiries which have led most clinicians to teach its importance in paresis, but by reason of the newer studies of Alzheimer on the character of the pathological alterations which show it to be an inflammation primarily involving the nervous parenchyma, and associated with vessel and neuroglia changes which point to a neurotoxic causation factor, but also by reason of the work of Wassermann and Plaut, and many others, who find that in the cerebrospinal fluid there is a peculiar substance that shows a definite reaction to syphilitic virus—a specific antibody—which seems to settle the matter. Just what the relations of this type of syphilitic poison may be to that of acute syphilis is still an unsolved problem, the factors of which I shall not even attempt to state. Contributory factors there may be, and these should not be overlooked. Not all syphilitics develop paresis. This is an important field in which many problems lie to be solved.

Before going any further, let us turn our attention to several cases which are here before us.

Case I.—W. S. This is a young Irishman, a clerk, married, with three healthy children, who has just come into the ward. He

has been in this country 20 years, and has always been an industrious and trusted employee in a large importing house.

You note in the first place that he is somewhat excited. His face lights up with a smile, and he laughs and jokes. His feet are tied with sheets, and he makes a few attempts to release them, and then sinks back, telling us with a smile and a laugh that it is a great joke.

He tells us that his father and mother died of old age, and laughs at the idea of there being any nervous disease in the family. He tells us he had measles and scarlet fever in childhood, but no other disease. He denies syphilis, yet you note a number of suspicious symmetrical copper colored spots on his body, which are very evidently tertiary syphilis.

In response to questions as to his whereabouts he says, "Why this is a hospital; I don't need to be here. I-I-I'm not s-s-ick. This is a joke. Why I've got a suit of clothes worth $35 and a watch, and you 've got me t-tied up here"—laughing—"why it 's a joke."

You note, as he tells me this, he does not seem to show any anger; he is constantly reaching down to loosen his feet and laughs. "I never felt better in my life. Why did they bring me here? I don't know. I think it was a joke." He tells us that he has been working as a cashier in Vessey Street. He remembers the number. Is not quite certain about the year of his birth, although he knows his age. He said he noticed that the bookkeeper made a mistake of $30 in the books, and that money was coming to him. He could give no definite reasons why he had left his place. In fact, he had been discharged some time past for drinking too much, and went to the office every day bothering them about the alleged mistake of $30 he had discovered in the books.

You note that he is approachable, is alert, fairly attentive. There is a monotony about his movements, however; they do not seem to be well directed; and he lies back every few seconds, with a grin, saying "it's a joke." You have probably noticed that there is a marked tremor of his hands as he sits there, and also that as he talks there is a definite tremor of the lips and of the tongue as he protrudes it. His speech, you observe, is slurring and stuttering at times. Note how he pronounces after me the words "National Intelligencer, Methodist Episcopal, Third Riding Artillery Bri-

gade." You note that on these test phrases he stumbles and stutters. Looking closer, you notice that one pupil is wider than the other, and that one is more sluggish than the other. His knee jerks are active, there is no clonus, no Babinski, no Oppenheim, no paradoxical reflex. You note that he is very voluble, but that he keeps saying the same thing over again; not in the verbigeration fashion of the precocious dement, but in a manner which shows he is distinctly dull or stupid, and somewhat hazy or confused. As you test his memory, you find he makes many slight blunders,—"7 x 6 = 42, 8 x 7 = 63, 7 x 8 = 56, 9 x 6 = 66, 6 x 9 = 54"—he makes these stupid mathematical blunders. The excitement might remind you of a press of activity of the maniac, if it were not so uniform and monotonous—badly executed. His speech is fairly voluble, but the association of ideas is not such as you have seen in the maniac phase of maniac-depressives; there is a loose association, which is more of a disconnected ramble.

We learn further, that this condition has developed slowly in a man 45 years of age; that he has been getting careless in his work; his relations with women have been becoming fairly loose; he began to drink rather heavily, coming to business about when he cared to; he was warned, but this doing no good, he was discharged. He pulled up a bit and was reinstated; once again to slip back into loose habits. In letters which he wrote to Ireland, he had planned some large enterprises. He was going to bring over his whole family, and they were going to do big things.

Just a few weeks before coming into the hospital, he was much agitated. His landlady says he used to walk the floor night and day, and that he would tear letters, papers, etc., into little bits of scraps and throw them out of the window, as though they were confetti. On being told not to make such a muss in the street, he promptly set fire to the little pieces, and threw them while burning into the street. He could not explain why he did this, although he denied indignantly that they had set fire to a curtain in a neighboring house—which was a fact.

Although he still retains a fair degree of intelligence, it is clear that there is a slight grade of defect.

This is an excellent example of a paretic in an early phase, and showing the physical signs more markedly, although indubitable

mental signs are not lacking. Up to the present time, he has shown no delusions unless one assume the $30 mistake in the books, to be one, and he has no hallucinations. He is excited, and, if subjected to restraint, is liable to become very violent. This is the type of case which, if let alone, may run for some time without much apparent deterioration, but when subjected to the restraint of an institution goes right up in the air, gets extremely violent, and is likely to die very shortly from exhaustion.

The next patient, to whom I wish to call your attention, presents features of even greater interest from the viewpoint of diagnosis. In the last case there is no excuse for any mistake in this regard. It is true that some physicians might call it a mania, but this would be due to lack of experience in mental diseases, or to careless observation. In this patient the correct appreciation of the disorder is not so apparent.

Case II.—J. E. B., 43 years of age; married. He is an Irishman, has been in this country since he was 6 years old, and is a policeman. He has one son living. He has always been a healthy man; he drank steadily, though not heavily, but gave it up about a year ago, as he felt it was doing him no good. He denies syphilis, and there are no visible signs of this disease on his body. He came to the hospital a few days previously of his own accord, saying he felt tired and needed a rest. He eats well, sleeps well at present, and feels that he has improved. He is well nourished and is apparently a strong man. Without going further into his history, let us see for ourselves what we can.

You note his manner is quiet and contained. He is placid and shows no signs of excitement or depression in his countenance. It is a little lacking in expression one might say, or denotes tire. His expression in general is not as vigorous as his size, age, and occupation might indicate.

In answer to questions he tells us that he is feeling better. He speaks you note in a quiet way, slowly and with a very slight retardation, *i.e.,* compared with the average of mankind. This may be his natural method of speech as he is not slower or more deliberate than many men, yet the slowness and quietness of manner are to be observed. He is quite particular in his enunciation, as though it were an effort for him to say things exactly as he would like, which

is suggestive of the neurasthenic reaction. You note on his repeating the test phrases that he says them correctly, and does not stumble, although they are pronounced in the precise, yet expressionless, or monotonous tones which characterize his whole speech.

Testing his motor power, we note a slight variation in the muscles of the face, the right side not having quite as much "life in it" as the left, yet there is no paralysis. The grasp, kick, push, etc., are equal and about up to a policeman's average. All of these movements, however, are made in a slow and deliberate manner, which is suggestive of the retardation of a maniac-depressive, but yet is not so marked. He has a slight tremor in his outstretched fingers, but it is not great.

His tendon reflexes are slightly exaggerated, but equally so; there is no ankle clonus, no Babinski, Oppenheim, or paradoxical reflex. There are no disturbances of sensation, save as we shall learn later. The pupils are dilated, at present they are slightly uneven, the right being a trifle larger than the left, but yesterday this was not the case, especially in the morning after he had had a good sleep. There is no Argyll Robertson pupil. No other neurological anomalies are obtainable.

He is well oriented as to time and place; his perceptions are normal. His association of ideas is a trifle superficial, and their time reaction slowed. His grasp is slow.

Asking him how his trouble came about he tells us that "Registry day, 1907, while on duty in front of the police station, he was leaning against a railing, and he kept this position for some time. When he stood up he noticed a numbness in his hands, which felt as though they were asleep. This went away, and he thought no more of it. He remembered he told his wife about it. He went back and was sent to another precinct. A short time later he noticed that his urine dribbled from him, or when he did urinate, the urine came in spasmodic bursts. He became nervous, could not sleep and had cold sweats. When he stopped work and went under treatment his urine troubled him no more.

He then noticed that the feeling of nervousness would come and go, in attacks as it were. They began, he said, in his head, leaving it numb; this went to his arm, and finally into the face. He could not talk for a nervous clutching in the throat, and while having

one of these feelings he was helpless. They soon passed off and left him very nervous."

This is the entire history up to the present time, and unless due stress be laid upon these paresthesiæ, the slow, almost dull flow of thought, the lack of initiative, the transitory bladder involvement, and the very slight irregularity in the pupils, one will put him down as a neurasthenic; perhaps even think that the peculiar clutchings at the throat are hysterical, yet this is undoubtedly a patient suffering from general paresis in the so-called neurasthenic phase which is so frequently a forerunner in this disease.

It is important constantly to bear in mind, whenever an adult about 40 years of age, comes to you suffering from the signs of a nerve tire or neurasthenia that there is a possibility of this disease, and a careful examination, going over the entire body, will repay your industry. I would call your attention particularly to the search for sensory anomalies, and ask you to be governed in your examination by the general rules laid down by Head in his Studies on Sensory Anomalies. You should test not only for light touch, for pressure, for pin prick, head and compass points, but particular stress should be laid on the bony conduction of sound vibration, on joint position and of pain due to deep pressure. Even in the earliest phases you may note some sensory disturbance that is more than can be accounted for by neurasthenia. In the present instance you note the marked sensory defects that this patient has to pain on deep pressure, especially in the arm in which he felt his paræsthesiæ. The monotony in this man's voice is also worthy of careful analysis. He has no true disturbance in his speech, no hesitation, no stumbling, no loss on the r's, p's, b's, yet you note that it takes some effort for him to get himself up to a good measure of performance. The vocal cords, however, are more than tired. The nervous regulation is being interfered with somewhat. It is not unusual to have the vocal cords affected very early. In singers, for instance, it is very striking, and not infrequently a diagnosis of paresis may be made first in an actor or singer, the giving out of whose vocal cords first calls attention to a disorder which has undoubtedly been present for some time.

Case III.—Now let me call your attention very briefly to this third case, which you can tell at a glance is one of general paresis.

He is 40 years of age, a carpenter; who was infected about fifteen years ago. He has been sick about one year, beginning in much the same ways as the disease in Case II, advancing at about six months to the stage which you have observed in Case I. We here find another phase of this many-sided psychosis. You noted with what a confident air he looks about; his whole bearing is one of confidence and self-satisfaction, and yet at the same time you can perceive that there is a slight inequality of the muscles of the two sides of the face, that he has a marked tremor about the muscles of the mouth and nasolabial folds, that his hands, as I ask him to stretch them out, have a distinct tremor, and that as he speaks his words stumble over each other very distinctly. He omits words, you will later observe. As I tell him what a fine muscle he has, you note how he feels it with satisfaction and says, "Yes, Doctor, I'm a strong man. Sullivan in his best days was nothing alongside of me." He goes on in a rambling manner, trying to tell you what he can do. Questioned as to whether he got the strength digging gold out of gold mines, he tells us how rich he is. What he is going to do with the million dollars a day that he gets; how he is going to start a fine establishment, and have two billion horses; his confidence in his various projects is unbounded, and they have such an absurd valuation that you recognize the typical megalomania of the paretic. Such megalomanias are often unbounded—anything you suggest he will take up and elaborate, all showing how well he will succeed, no matter what others have done before him. There is a certain lack of interest in what he says just now—he has none of the brightness of a patient in the maniacal phase, although he shows a certain amount of suggestibility in taking up ideas, a very characteristic divertability, and at times a loose ramble of ideas which suggests flight of ideas, and in some patients with paresis may be a true flight, just as in a manic; but his very obvious memory defects, his physical signs, his megalomania are too characteristic to admit of question.

This morning he was in a wild state of excitement: he was shouting and noisy, and for two days has been soiling himself. About three weeks ago he had an epileptiform convulsion and was hemiplegic for about three or four days. This has disappeared now, yet you note a difference in the facial innervation, his grip is unequal, and although both tendon reflexes are active that of the left

side is more marked. He shows some clonus on that side as well.

This leads me to say a word about these epileptiform or apoplectiform convulsions, which are not infrequent in general paresis. It is occasionally observed that almost the first sign known to a patient's uncritical environment will be a convulsive seizure, from which the patient usually recovers in three or four days with little trace of any difficulty. These early attacks are unusual, but occur with sufficient frequency not to be forgotten. In the course of a paresis, especially if it be at all active, convulsive phenomena either epileptiform, or apoplectiform, will occur in at least one-half of the cases before death, and such attacks are often the cause of death. It is noteworthy to observe, notwithstanding the extreme severity of the convulsive attack, the hemiplegia, etc., that it will usually clear up in a comparatively short time. Occasionally we find permanent atypical localized lesions (Lissauer's type).

This type of case, which is spoken of as the classical type of paresis, is worthy of careful examination. We are told by many that this type is changing; that the demented and simple depressed types are becoming more frequent. This I am unable to believe, and feel that what has changed is, not the disease,—this has been affecting minds of much the same type for thousands of years—but our clinical insight into the many vagaries of the disorder has been greatly sharpened and that while thirty years ago only those who showed the megalomania, etc., were thought to have paresis, we now recognize that the grouping of symptoms is largely accidental and non-essential, hence the quiet, slow dementing, and depressed paretics now enter into statistics under the proper cognomen.

Case IV.—Now let me call your particular attention to this young man, 38 years of age, a reporter, who appears to be perfectly well. I wish you to talk to him and examine him individually, for when I say that he seems perfectly well, and not one physician in a thousand would suspect that he was a sick man, you will be astonished at what I shall tell you concerning him.

He is perfectly oriented, his memory is perfect, he acknowledges syphilis thirteen years previously, also alcoholism, not marked. His speech is slightly rapid, but this is his usual manner. He is bright, energetic, and for six months has been carrying on a successful business as a real estate agent, although he is not working very hard.

His pupils are equal and react to light and accommodation; he has no tremor. There is slight loss of knee jerks and he says he feels like a trivet, and is as sound as he ever was.

Search where you will and you will be unable to find a trace of illness in him, and yet, one year ago this young man, whom we have just excused, was a bedridden, apparently hopelessly demented paretic, worse than any you have yet seen. He had unequal pupils; had a stammering, stuttering speech, and was worth millions. He expressed delusions of all fantastic characters, was megalomaniac, hallucinatory, and violent. He soiled his bed, and had three severe apoplectiform convulsions. This condition was of gradual onset, persisted about six months, and a month before he began to recover he had his third epileptiform attack—he was dumb, almost moribund, and did not know anything. He gradually picked up, and after a month was able to be about. In two months he went home, and is now, nine months later, in the condition in which you have just seen him.

This patient is in what is known as a "remission." In some unknow manner the acute, productive inflammation in his brain tissues has subsided, and has left him comparatively well, but,—and here is the important lesson to be drawn from the case—if the diagnosis is correct, and we have not mistaken a case of cerebrospinal syphilis, or acute alcoholic dementia with epileptiform convulsions for paresis—and this is possible—this patient will again be sick, and will ultimately die of general paresis. In this case a lumbar puncture showed the typical lymphocyte count, thus excluding alcohol as an exciting cause—it may be a case of cerebrospinal syphilis, and most of the so-called *cured* cases of paresis are this disease; but the general development of the disorder has been such as to make us feel that it is a case of paresis. Look out for these remissions then, and govern yourselves accordingly. They sometimes last as long as two years, although six to ten months is the average.

Varieties.—A brief word as to types. In our pathological study we have seen why so varied a clinical picture may be present; yet the cases as they are seen follow certain lines,—(1) demented forms, which go slowly onward; (2) depressive forms, like our Case II; (3) expansive or classical forms, like Case III, and (4)

agitated forms, like Case I, are the most usual. It should not be forgotten that the picture changes from week to week in the vast majority of cases, and only a few cases will run by the set conventions of a textbook description.

Course.—The course of the disease is uniformly fatal. In some patients death takes place within comparatively few months; while other patients are known who have lived, 10, 12, 14, and even 21 years.[1]

The most recent and available statistics on this interesting question may be found in an analysis of over 3,000 cases in the Berlin Asylum of Dalldorf by Junius and Arndt (Arch. f. Psych., vol. 44, heft. 1, 1908).

It is established that few patients suffering from general paresis live over six years after the onset of their illness; in the cases cited, less than one per cent. of the 3,400 cases lived over six years. A great many (about 19 per cent.) died within the first year, about 25 per cent. in the second year, 25 per cent. in the third year, 7 per cent. in the fifth year and from 7 to 10 per cent. lived over five years.

Differential Diagnosis.—In what I have said on pathology, I have clearly indicated to you what the important features are which enter into the question of differential diagnosis. It will not be necessary for me to repeat them at this time, and I shall ask your strict attention to the different disorders, the pathology of which I have detailed, and the symptomatology which it is necessary to bear in mind in order to exclude these factors.

Treatment.—From one point of view there is no treatment for paresis, yet at the same time this only expresses the general attitude towards the possibility of remedial agencies in the cure of the condition. There is a great deal that can be done for the paretic individual to make him more comfortable, and to remedy the disturbances and distress in the household of which he is a member.

It is in the first place, I believe, absolutely essential that as soon as a diagnosis is made a proper representative of the interests of the sick individual should be carefully instructed as to the probable outcome of the condition. You should always bear in mind, therefore, the possibility that you can make a mistake in the diagnosis,

---

[1] Kraepelin, Behr, Sprengeler, Heilbronner, Kaes, Lustig.

and should therefore reserve for yourselves the right of a definite standpoint. The property interests should be conserved at once, for it is very characteristic that a paretic dissipates his fortune in riotous living, and in large communities particularly, is preyed upon by numberless human parasites, male and female, who infest such communities.

Careful notes should be taken and kept by you all, because very important medico-legal problems may later arise in the matter of the making of wills, deeding of property, etc., by such sick individuals as a result of their weakened powers of resistance, and their hypersuggestibility when surrounded by self-seeking interests.

The treatment in these cases is essentially an economic and social one, and your responsibility in this respect should be carefully considered. These patients should be watched very carefully if there is any suspicion whatever of cerebrospinal syphilis, and such a suspicion we have already seen can never be entirely eliminated. Prompt mercurial treatment should be instituted. From my own point of view I am very skeptical on the question of the possible relationship of mercurial poisoning and cerebral degeneration, so that prompt antisyphilitic treatment is not calculated to do any harm whatever.

It is advisable, in practically all instances, to have these patients immediately adjudged incompetent and to have them placed in some private or public hospital where they can be taken care of. It is useless, as a rule, to attempt to treat them at home. In the neurasthenic phases it may be necessary to use hypnotics, to assist sleep; it may be essential to give analgesics to overcome pain; it may be desirable to give bromides to quiet excitement. For the treatment of excitement which is excessive there is nothing so valuable as the prolonged bath, which is far better than any other mode of of treatment.

Restraint, chemical or physical, usually acts disastrously on the paretic, tending to excite him, and to hasten the exhaustion which brings about death.